

Ref #	Hits	Search Query	DBs	Default Operator	Plurals	Time Stamp
L1	18	Ruvkun NEAR Gary	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/10/04 13:21
L2	827	PTEN	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/10/04 13:22
L3	15	daf-18	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/10/04 13:22
L4	453	L2 and (obesity glucose)	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/10/04 13:22
L5	111	lipid phosphatase	US-PGPUB; USPAT; EPO; JPO; DERWENT	NEAR	ON	2005/10/04 13:23
L6	66	L2 and L5	US-PGPUB; USPAT; EPO; JPO; DERWENT	NEAR	ON	2005/10/04 13:23
L7	24	L5 and obesity	US-PGPUB; USPAT; EPO; JPO; DERWENT	NEAR	ON	2005/10/04 13:24
L8	3	pten lipid phosphatase obesity	US-PGPUB; USPAT; EPO; JPO; DERWENT	SAME	ON	2005/10/04 13:24
L9	4	pten lipid phosphatase glucose	US-PGPUB; USPAT; EPO; JPO; DERWENT	SAME	ON	2005/10/04 13:24
L10	3	pten phosphatase obesity	US-PGPUB; USPAT; EPO; JPO; DERWENT	SAME	ON	2005/10/04 13:25
L11	64	pten phosphatase and obesity	US-PGPUB; USPAT; EPO; JPO; DERWENT	SAME	ON	2005/10/04 13:25
L12	6	ogg scott	US-PGPUB; USPAT; EPO; JPO; DERWENT	NEAR	ON	2005/10/04 13:25
L13	4	l12 and l2	US-PGPUB; USPAT; EPO; JPO; DERWENT	NEAR	ON	2005/10/04 13:26

L14	6	I12 and I1	US-PGPUB; USPAT; EPO; JPO; DERWENT	NEAR	ON	2005/10/04 13:26
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=> d his

(FILE 'HOME' ENTERED AT 13:28:10 ON 04 OCT 2005)

FILE 'MEDLINE, CANCERLIT, AGRICOLA, CAPLUS, SCISEARCH' ENTERED AT  
13:28:18 ON 04 OCT 2005

L1 7018 S PTEN  
L2 774 S LIPID PHOSPHATASE  
L3 180319 S OBESITY  
L4 14460 S IMPAIRED GLUCOSE  
L5 0 S L1 (L) L2 (L) L3 (L) L4  
L6 447 S L1 (L) L2  
L7 0 S L6 (L) L3  
L8 1 S L6 (L) L4  
E RUVKUN GARY?/AU  
E RUVKUN G?/AU  
L9 176 S E1  
E OGG S?/AU  
L10 11 S E4  
L11 187 S L9 OR L10  
L12 5 S L11 AND L1  
L13 2 DUP REM L12 (3 DUPLICATES REMOVED)  
L14 35 S L1 AND L3  
L15 25 DUP REM L14 (10 DUPLICATES REMOVED)  
L16 1 S L1 AND L4  
L17 0 S L15 AND PY<=1997

=> d an ti so au ab pi l16

L16 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2005 ACS on STN  
AN 2000:384548 CAPLUS  
DN 133:39116  
TI Genes and polypeptides involved in insulin signaling pathways for glucose tolerance, obesity, and longevity and their uses as therapeutic and diagnostic tools  
SO PCT Int. Appl., 402 pp.  
CODEN: PIXXD2  
IN Ruvkun, Gary; Ogg, Scott  
AB Disclosed herein are novel genes and methods for the screening of therapeutics useful for treating **impaired glucose** tolerance conditions, as well as diagnostics and therapeutic compns. for identifying or treating such conditions. The *Caenorhabditis elegans* metabolic regulatory genes *daf-2* and *age-1* encode homologs of the mammalian insulin receptor/phosphoinositol 3-kinase signaling pathway proteins, resp. Also, the *C. elegans* PKB kinase and AKT kinase act downstream of these genes, as their mammalian homologs act downstream of insulin signaling. The *C. elegans* **PTEN** lipid phosphatase homolog, *DAF-18*, acts upstream of AKT in this signaling pathway. Further, the *DAF-16* forkhead protein represents the major transcriptional output of this insulin signaling pathway. Addnl. evidence indicates that the *DAF-16*, *DAF-3*, *DAF-8*, and *DAF-14* transcriptional outputs of converging signaling pathways regulate metabolism. The congruence between the *C. elegans* and mammalian insulin signaling pathways strongly supports the contention that new genes identified in the *C. elegans* pathway also act in mammalian insulin signaling. Exemplary sequences and functional characteristics of the *C. elegans* *daf* genes and their human homologs are provided.  
PATENT NO. KIND DATE APPLICATION NO. DATE  
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PI WO 2000033068 A1 20000608 WO 1999-US28529 19991202  
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM  
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG  
US 2001029617 A1 20011011 US 1998-205658 19981203

US 6861256                    B2       20050301  
AU 2000017496                A5       20000619       AU 2000-17496               19991202  
EP 1163515                    A1       20011219       EP 1999-960641               19991202  
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,  
IE, SI, LT, LV, FI, RO

L6 ANSWER 2 OF 9 MEDLINE on STN DUPLICATE 2  
AN 2004545479 MEDLINE  
TI Enhanced insulin sensitivity, energy expenditure and thermogenesis in  
adipose-specific Pten suppression in mice.  
SO Nature medicine, (2004 Nov) 10 (11) 1208-15. Electronic Publication:  
2004-10-17.  
Journal code: 9502015. ISSN: 1078-8956.  
AU Komazawa Nobuyasu; Matsuda Morihiro; Kondoh Gen; Mizunoya Wataru; Iwaki  
Masanori; Takagi Toshiyuki; Sumikawa Yasuyuki; Inoue Kazuo; Suzuki Akira;  
Mak Tak Wah; Nakano Toru; Fushiki Tohru; Takeda Junji; Shimomura Iichiro  
AB **Pten** is an important phosphatase, suppressing the  
phosphatidylinositol-3 kinase/Akt pathway. Here, we generated  
adipose-specific **Pten**-deficient (AdipoPten-KO) mice, using newly  
generated Acdc promoter-driven Cre transgenic mice. AdipoPten-KO mice  
showed lower body and adipose tissue weights despite hyperphagia and  
enhanced insulin sensitivity with induced phosphorylation of Akt in  
adipose tissue. AdipoPten-KO mice also showed marked hyperthermia and  
increased energy expenditure with induced mitochondriogenesis in adipose  
tissue, associated with marked reduction of p53, inactivation of Rb,  
phosphorylation of cyclic AMP response element binding protein (CREB) and  
increased expression of Ppargc1a, the gene that encodes peroxisome  
proliferative activated receptor gamma coactivator 1 alpha.  
Physiologically, adipose **Pten** mRNA decreased with exposure to  
cold and increased with **obesity**, which were linked to the mRNA  
alterations of mitochondriogenesis. Our results suggest that altered  
expression of adipose **Pten** could regulate insulin sensitivity  
and energy expenditure. Suppression of adipose **Pten** may become  
a beneficial strategy to treat type 2 diabetes and **obesity**.